CA1 Mini PBLD 22.3: COPD Patient Ventilator Management

Authors:

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Learning Objectives:

Upon completion of this learning activity, participants will be able to:

- Discuss the pathophysiology of COPD.
- Describe the basic considerations and treatments in individuals suffering from COPD.
- Discuss appropriate ventilator management for patients with COPD.

Case: A 68 year old man with COPD just underwent a laparoscopic cholecystectomy (under general anesthesia). Following the procedure, the patient was extubated in the operating room and was transported to the post-anesthesia care unit (PACU) with supplemental oxygen (2L nasal cannula). During his time in the PACU, the nurse notes the patient is struggling to breathe and he continues to require supplemental oxygen (he does not require supplemental oxygen at home). Upon arrival to the PACU, you note the patient's oxygen needs have increased significantly (now on 60% FiO_2 via high-flow nasal cannula) and his respiratory rate is 25-30 breaths per minute.

1) What is COPD?

- *COPD (Chronic Obstructive Pulmonary Disease) is a common condition characterized by the obstruction of airway flow. According to the Global Initiative for Chronic Obstructive Lung Disease (GOLD),COPD is defined as follows:*

  - *"COPD is a common, preventable, and treatable disease that is characterized by persistent respiratory symptoms and airflow limitation that is due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases. The chronic airflow limitation that characterizes COPD is caused by a mixture of small airways disease (e.g., obstructive bronchiolitis) and parenchymal destruction (emphysema), the relative contributions of which vary from person to person. Chronic inflammation causes structural changes, small airways narrowing, and destruction of lung parenchyma. A loss of small airways may contribute to airflow limitation and mucociliary dysfunction, a characteristic feature of the disease."*

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- *In short, COPD is a chronic inflammatory lung disease that causes obstructed airflow from the lungs.*

2) What other causes of respiratory distress besides COPD are in your differential diagnosis?

- *Residual Medication Effects (excessive opioids, residual muscle relaxant)*
- *Myocardial Infarction*
- *CHF/Fluid Overload/Pulmonary Edema*
- *Obstructive Sleep Apnea*
- *Aspiration Event*
- *Pulmonary Embolism*
- *Atelectasis*
- *Residual CO_2 from Abdominal Insufflation*
- *Splinting from inadequate Pain Control (inability to take deep breaths and adequately cough)*
- *Pneumothorax.*
- *Pneumonia.*

3) You suspect the patient's COPD is playing a significant role in the patient's respiratory distress, but you are aware there could be other factors as well. What steps would you take to investigate the cause of the patient's respiratory distress?

- *The assessment should begin with a directed bedside physical exam (although studies can also be ordered while you are beginning your initial assessment.) Diagnostic studies should also be considered to assess for the cause of the patient's distress (see below). The patient's anesthesia record should also be reviewed along with the patient's medical history to assess for possible residual drug effects and fluid overload.*

- *Vital signs: BP, HR, RR, oxygen saturation, temp*
- *Physical Exam:*
  - *Visual Evaluation of the patient's breathing pattern.*
  - *Evaluation of the patient's airway for possible obstruction.*
  - *Auscultation of the patient's heart and lungs.*
  - *Evaluation of volume status for possible volume overload (peripheral edema, JVD).*
  - *Assessment of vital signs and amount of oxygen/ventilator support.*
  - *Evaluation of the patient's level of consciousness.*
- *Laboratory Studies:*
  - *Arterial Blood Gas.*
- *Imaging Studies/Other Studies:*
  - *Portable Chest X-ray.*
  - *Electrocardiogram.*

- *Echocardiogram (bedside transthoracic echo by either you or the cardiology service) to evaluate for cardiovascular function, volume status, and pulmonary status (alveolar interstitial syndrome, pneumothorax, pleural effusion)*
- *Train-of-Four Evaluation (for adequate muscle relaxant reversal evaluation).*

4) What findings in evaluation of the patient would be most consistent with an exacerbation of COPD?

- *Physical Exam: The patient is likely awake and in visible respiratory distress. SpO_2 would be decreased despite the use of supplemental oxygen and his respiratory rate would be increased. On auscultation, wheezing may be heard (although this may be absent is severe exacerbations). There would not be signs of upper airway obstruction (for example, snoring). With severe hypercarbia (greater than 90 mm Hg) mental status could be depressed.*
- *Laboratory Studies: An ABG would likely show some degree of hypoxemia and hypercarbia.*
- *Imaging/Other Studies: The patient's chest x-ray would likely not show obvious pulmonary edema or infiltration but rather hyperinflated lungs (possibly with a flattened diaphragm, see image below).*

Image courtesy of Dr. Jeremy Jones, Radiopaedia.org.

5) **Following your investigation, you feel confident the patient is suffering from an exacerbation of his COPD. How would you manage and treat this patient?**

- *Of note, COPD exacerbations typically are started by a type of "trigger." Common triggers may include infectious etiologies or an aspiration event.*
- *Stabilization: The first step should be to stabilize the patient (ensure the patient is able to adequately oxygenate and ventilate while pharmacologic treatment is initiated).*
- *Pharmacologic treatments: Beta adrenergic agonists, anticholinergic agents, glucocorticoid therapy, and antibiotics (if infectious etiology). Beta-adrenergic agents stimulate β (particularly β_2) receptors on bronchial smooth muscle causing adenylyl cyclase to convert ATP to cAMP which results inhibition of myosin phosphorylation and lowering of intracellular calcium which ultimately results in bronchial smooth muscle relaxation (reducing obstruction and improving airflow). Anticholinergic agents block muscarinic acetylcholine receptors which promotes the degradation of cGMP and this in turn decreases contractility of smooth muscle in lung (reducing obstruction and promoting better flow). Anticholinergic medications also reduces mucus secretion. Glucocorticoid medications (both inhaled and intravenous) reduce inflammation of the airway by way of their steroidal activities. The beta adrenergic and anticholinergic agents will be your most rapidly acting treatments to acutely manage the patient, but initiation of other treatments should be initiated if a COPD exacerbation is suspected.*
- *Supportive Care: Oxygen therapy, non-invasive positive pressure ventilation (NIPPV), mechanical ventilation. Transition from just supplemental oxygen to non-invasive positive pressure may be prompted by worsening oxygenation, worsening hypercarbia, or increased work of breathing. Transition from NIPPV to traditional mechanical ventilation my be sought due to continued poor oxygenation and continued CO_2 retention despite NIPPV.*

6) **Despite your efforts with support and pharmacologic treatments, the patient's respiratory status continues to worsen, and the patient requires mechanical ventilation. What are the ventilator strategies that need to be considered when providing mechanical ventilation for a patient with COPD?**

- *When a patient with COPD (especially in an exacerbation) requires mechanical ventilation, remember the two primary pathophysiologic issues that contribute to acute respiratory failure in COPD: increased airway resistance and dynamic hyperinflation.*
- *The ventilator strategies in such a patient has two aims: correcting the gas exchange issue and identifying/preventing dynamic hyperinflation.*
    - *Correction of gas exchange can be achieved by allowing adequate time for exhalation, this can be accomplished by allowing an increased proportion of time for exhalation. It is not uncommon for patients with COPD to require an inspiratory time: expiratory time ratio (I:E ratio) of 1:3 to 1:5. Optimization of sedation may allow for better synchronization with the ventilator.*

- *Dynamic hyperinflation should be suspected if airway pressures progressively rise and/or if it appears that each breath is not being entirely expired. Severe hyperinflation can lead to hemodynamic compromise and even pneumothorax.*
- *Preventive measures against hyperdynamic inflation include avoiding excessive minute ventilation (reductions in tidal volume and respiratory rate to allow more time for expiration), adjustment of I:E ratio (see above), and selection of appropriate ventilator mode.*

- *Overall goals of ventilation:*
  - *Minimize airway pressures.*
  - *Minimize work of breathing*
  - *Maximize expiratory time for each breath delivered.*
  - *Avoid excessive ventilation (over correction of CO_2).*
  - *It should be noted, in those with COPD exacerbations, mechanical ventilation doesn't solve the problem, it only supports ventilation while the exacerbation is treated. The goal should always be prompt recognition and treatment of the underlying cause.*

7) What is "auto-PEEP?"

- *This is also known as intrinsic peep. This occurs when ventilator settings produce an I:E) ratio that does not allow adequate time for expiration. This means a subsequent breath is delivered prior to complete emptying from the previous breath. This gas trapping can cause significant elevations in end-expiratory pressure, leading to increases in airway pressures and intrathoracic pressure. Hypotension and barotrauma (causing pneumothorax) are potential complications.*
- *Because COPD increases the required expiratory time, mechanically ventilated patients are at high risk for auto-PEEP. The most effective way of avoid/combating auto-PEEP is increasing the actual time for expiration made available for each breath. Optimization of ventilatory synchrony, avoidance of excessive minute ventilation, and appropriate ventilator mode selection are also important considerations.*

8) What ventilator strategy would you employ if the initial ABG comes back with
pH – 7.23
$paCO_2$ – 81
paO_2- 89
Bicarb – 35

- *Many individuals with COPD are chronic retainers of CO_2. Mechanisms of CO_2 retention in these individuals include the disruption of capillary beds, airway obstruction (leading to hyperinflation), and reduced lung compliance all leading to an increase in dead space (V/Q mismatch). This chronic increase in CO_2 will cause a reduction in the patient's pH but this is compensated by the renal system (the kidneys "hold on to" more or "dump" less bicarbonate. With this in mind, one should aim to normalize the patient's pH, not*

the $paCO_2$. Normalization of CO_2 in someone with a chronic respiratory acidosis can cause a severe alkalemia. Furthermore, a prolonged time on the ventilator with this correction may cause the kidneys to compensate and removed additional bicarbonate making weaning from the ventilator much more difficult.

Suggested Readings:

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